FOXP2: A gene of linguistic importance

Stephanie Hunt
Bryn Mawr College

The gene *FOXP2* was discovered to be mutated in members of a family possessing a severe language disorder. Originally some researchers believed that this gene was responsible for grammar and called it the "language gene". After further study of the family, researchers determined that the gene itself was not generally responsible for grammar but that it may be responsible for the development of structures required for spoken language. The present paper suggests that *FOXP2* was played a role in the emergence of spoken language during human evolution. This is supported by evidence from human and nonhuman primates and the study of the gene itself. Proposed accounts of how *FOXP2* may be related to the emergence of language are given. Furthermore, the present paper hypothesizes that *FoxP2* may be causally related to the "critical period" for language acquisition. This hypothesis is supported by evidence from vocal-learning birds, specifically that the levels of the *FoxP2* protein vary depending on whether the birds are learning new songs during that time. Furthermore, a very brief discussion of how *FOXP2* may be related to symptoms of schizophrenia is included in order to provide a comprehensive account of the gene's relationship to language. Finally, discussed are the ethical issues surrounding the study of *FoxP2*'s relationship to the critical period for language acquisition.[*]

Language may be only one faculty of many that sets humans apart from other creatures, but it may be the crucial one in humans' domination over other creatures on Earth. While nonhuman animals display methods of communication, human language is much more complex than any other known system of communication in the animal kingdom. Could humans' superior language abilities be the so-called "X-factor" that has set humans apart from other animals and has led to our nearly complete control over many animals on Earth? If so, one might question how human language came into existence in *homo sapiens*, but did not emerge in any other species of primates. One place to search for the answer to this question is within the field of genetics. A gene

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called *FOXP2* (an acronym for "forkhead box P2") has been linked to linguistic communication. The gene is also believed to have made its most recent mutation, which resulted in its current state in humans, at approximately the same time that human vocal language emerged. Although *FOXP2* was originally described as "the language gene" in the popular media when it was first discovered, one should note that the gene is not the sole factor through which humans pass language on to their offspring. While *FOXP2* may not be the "language gene" that it is sometimes claimed to be, it may be the gene that led to language during human evolution, since there is evidence of genetic selection of the gene during a time that was crucial to the development of vocal communication.

Another aspect of human language that may be influenced by *FOXP2* is the critical period for language acquisition during childhood in humans. The levels of *FOXP2* in language specific areas of the brain at times when language acquisition is occurring may be responsible for this specific, critical period required for the learning of vocal communication. The connection between levels of the *FoxP2* protein and the acquisition period for vocal communication has already been validated in the brains of vocal-learning birds. It may be the case that *FoxP2* in some way causes plasticity, or malleability, which is required for learning, in the particular areas of the brain associated with vocal learning. Thus, a second result of *FOXP2*'s presence in vocal learners may be that it enabled the learning of language at developmentally appropriate periods throughout the lifespan.

The present analysis draws on previous research in a variety of areas related to *FoxP2* and the evolution of language to demonstrate that there is a high probability of *FoxP2*'s relationship to the emergence of vocal communication upon the human lineage.

It is claimed that this relationship allowed for the ability of humans to physically engage in vocal communication. Furthermore, the hypothesis is made that *FoxP2* is potentially responsible for the critical period of language acquisition. A brief summary of research regarding *FOXP2's* possible connection to schizophrenia is also included in order to provide a full analysis of what is presently known about the gene.

DISCOVERING *FOXP2*

In 1990, a large family in England known as the KE family was discovered to have an inherited language disorder known as Specific Language Impairment (SLI)[†] (Vargh-Khardem et al. 2005). The distribution of the disorder suggested to researchers that it was caused by a dominant gene on an autosomal chromosome: half of the family members are affected by the disorder and half are not. Figure 1 (below) shows the pedigree of the KE family:

Figure 1

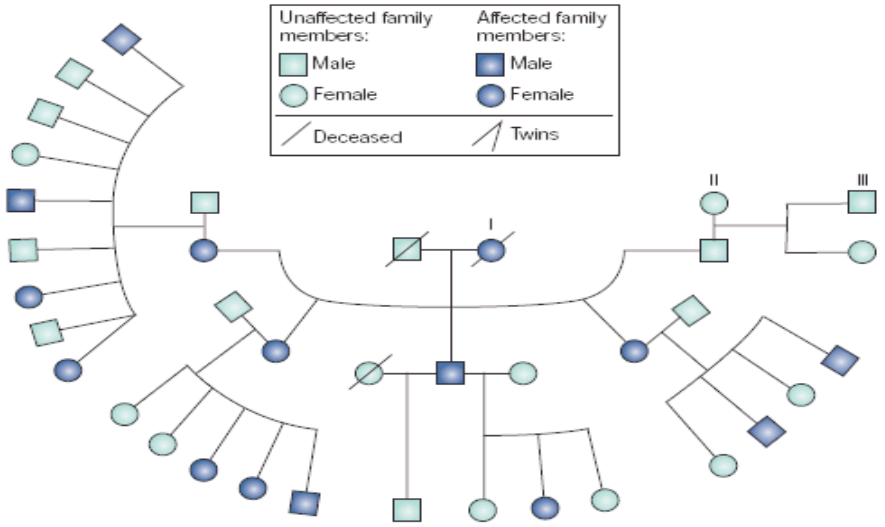

Figure 1 | **Pedigree of the KE family.** I, II and III represent the generations. Modified, with permission, from REF. 14 © (2002) Oxford University Press.

[†] It should be pointed out that the disorder Specific Language Impairment is not necessarily genetic, although there is evidence that it may sometimes be hereditary based on studies of affected children with positive family histories of SLI (Ahmed, Lombardino, & Leonard 2001).

from Vargha-Kardem et al. 2005.

However, even with a family with such a simple single-gene disorder, which is unusual since many traits are caused by multiple genes, it was not easy to track down the specific point of mutation. In order to do this, geneticists had to look at the distribution of a set of markers, or small pieces of DNA with a specific location, that vary between individuals. They searched for a correlation between the distribution of markers and the distribution of affected family members. The region that was indicated by this search was a locus on the long arm of chromosome 7, informing the researchers to look deeper into this area of about 70 genes for the disruption. Another individual known as C.S. with a disorder similar to that of the KE family was discovered to have a chromosomal rearrangement. Part of chromosome 7 had actually broken off and attached itself to another chromosome in C.S.'s DNA. The point at which the chromosome had broken was right in the middle of the implicated area of the disruption in the KE family. Analysis of the breakpoint showed that the gene at that point was part of a group responsible for encoding *forkhead transcription factors*, which produce proteins, and hence was named *FOXP2*, which stands for Forkhead bOX P2, where *P* represents the branch of the *FOX* (forkhead box) family and *2* indicates that it was the second gene in branch *P* to be found (Marcus & Fisher 2003).[‡,§] *FOX* genes are explained in greater detail below. Once the exact location of the gene in question had been found, *FOXP2* in the KE family was analyzed. Indeed, the affected family members had an inherited change in a single nucleotide (groups of molecules that are building-blocks for DNA) causing a disruption in the functioning of

[‡] *FOXP2* (in capital letters) represents the gene itself in humans, but in lowercase letters (*FoxP2*), it refers to the gene in non-human animals. When it is not italicized, (FOXP2 or FoxP2), it refers to the expression or phenotype of the gene (Cooper, 2006).
[§]Below, I will discuss the fact that *FoxP1* may play a role in vocal behavior as well (Scharff & Haesler 2005; Scharff & White 2004).

the protein produced by *FOXP2* (Marcus & Fisher 2003). A single guanine nucleotide is replaced with an adenine nucleotide (Pinker 2001). The pattern of the mutation taken with the deficits of the affected family members is evidence that the genetic mutation is correlated with the Specific Language Impairment experienced by these individuals.

Although it is now known that the change in the amino-acid in *FOXP2* is correlated with the language difficulties of the KE family, it is still undetermined as to how exactly the gene is causing the problems. Initially, the disorder was referred to as "developmental verbal dyspraxia" which is characterized by trouble with articulation (Marcus & Fisher 2003). Following the news of the discovery of the KE family a number of different hypotheses about the specific nature of the disorder were made. Some characterized the disorder as a dysphasia or an inability to use the rules of English morphology for denotation of tense and number (Gopnik & Goad 1997). Other researchers believed it was a problem with phonological and language production systems, while yet others simply categorized it as a severe speech disorder across all aspects of language (Vargha-Khardem et al. 2005).

One interpretation of the disorder, and thus, the function of the gene, is that the mutation in the gene causes grammatical impairments in both expressive and receptive language (Bishop 2002). The main proponent of the theory that genetics influences grammar itself is Myrna Gopnik (Gopnik & Goad 1997). Gopnik and Goad classify the KE family into a group of people with a genetic dysphasia. These authors say that people with a genetic dysphasia lack morphological markers in their underlying grammar (1997). When Gopnik started making this claim about the KE family and that the gene influenced grammar, it was quickly picked up by the mainstream media. A simple Internet search

brings up articles from the media declaring *FOXP2* to be a "language gene" and misleading titles asserting that the "language gene" has left apes speechless and that humans share the "language gene" with birds. While these assertions may have some basis in scientific evidence, they imply to the incompletely informed reader that *FOXP2* is the sole gene that is responsible for the language capacities of humans. After further research on the KE family, it has been made clear that *FOXP2* does not simply influence a person's underlying grammar.

Deficits of the KE family

Vargha-Kardem and colleagues (2005) tested the members of the KE family in numerous language tasks and other cognitive measures. Although they did find that affected family members do have some problems with articulating phonemes and controlling oro-facial movements, they also have trouble identifying basic speech sounds spoken aloud to them, understanding sentences, and judging grammaticality of phrases. Thus, the problem is not simply with motor control, as one may think upon first glance at the KE family, because if this were the case, only deficits in production would be found. Another major finding was that the language disorder is not simply caused by a low IQ score, another potential "first glance" explanation of the KE family's deficits. The IQ scores of affected family members were within the normal range and, in some cases, even higher than unaffected family members (Marcus & Fisher 2003).

Affected family members also had difficulty repeating polysyllabic words compared to monosyllabic words (Vargha-Khardem et al. 2005). Voxel-based morphometry scans, which are used to identify specific differences in brain matter between groups of scans by comparing small sites called *voxels*, were performed on the

KE family as well. The scans showed that the neuropathology involves multiple components of the motor system and is bilateral. Thus, since the pathology was found in both hemispheres of the brain, there was not a drastic reorganization of contralateral pathways in the brain, which allowed the affected members to retain basic speech abilities. A positron emission tomography (PET) scan showed that both of the caudate nuclei in the affected family members were reduced by about 25%. The size of the caudate nuclei, parts of the brain important to learning and memory systems, significantly correlated with the individual's score on both the oral praxis (a test of non-word repetitions) and the coding subset (a test of visual-motor coordination) of the Wechsler Intelligence Scale. This finding suggests a relationship between the caudate nucleus and the oromotor control and articulation difficulties in the affected family members (Vargha-Khardem et al. 2005).

Not only were affected members of the KE family deficient in many areas of language and oromotor control, there were also general deficiencies in their perception of timing (Alcock et al. 2000). This was found by testing them on musical patterns—affected individuals performed normally on tasks of pitch perception but had significant difficulty in extracting rhythm from music and producing rhythm. This finding is common to most people with SLI (Alcock et al. 2000). As this task does not require any oromotor ability, the results cannot be explained by the affected family members' deficiencies in this area. However, a possible explanation is that some brain areas required for language interpretation and production also detect and produce rhythm (Alcock et al. 2000). This makes sense, as normal conversation could be significantly

more difficult for people who have trouble detecting and producing rhythm in speech, as it is for the affected members of the KE family.

Furthermore, Vargha-Kardem and colleagues found that the affected members of the KE family had activation in parts of their brains during generation of verbs and repetition tasks that are not usually activated by these tasks (2005). Unaffected family members showed a normal left-dominant pattern of activation, which involved Broca's area, a known speech center in the left-hemisphere. The affected family members showed a more posterior and more bilateral pattern of activation in these tasks. Moreover, affected members showed a general pattern of over-activation in other regions of the brain not usually associated with language. This pattern could exemplify compensation of the brain for the circuits that might not have developed to properly accommodate language abilities. It could also simply be reflective of extra cognitive effort involved in performing verbal tasks for these individuals. These findings taken together demonstrate that *FOXP2* plays a role in development of networks in the brain that are involved in learning, planning and executing speech motor sequences (Vargha-Khardem et al. 2005). Yet another possibility regarding the pattern of activation of the affected KE family members, brought up by Corballis (2004), is that this pattern may implicate *FOXP2* in the lateralization of Broca's area for speech. However, as Corballis points out, this idea should be taken skeptically as it would have serious ramifications on the notion of lateralization generally. There is no evidence that *FOXP2* controls lateralization of the brain, however, as will be discussed below, *FOXP2* may have influenced Broca's area in other ways during evolution (Corballis 2004).

FUNCTIONS OF *FoxP2* AND OTHER *FOX* GENES

As previously mentioned, there have been numerous theories about what *FOXP2*'s actual function is. To begin with, and simply as a point of interest, *FOX* genes are only found in animals and fungi and studies show that there is a direct correlation between the number of different *FOX* genes and the neural complexity of the organism. For example, a yeast genome has four different *FOX* genes, a nematode has 15, a fruit-fly has 20 and humans have at least 40. *FOX* genes have a known importance in the development of embryos, and therefore, it is likely that a higher number of distinct *FOX* genes are necessary for development depending on the complexity of physical structures in the body (Marcus & Fisher 2003). They are related to a number of various developmental disorders (Cooper 2006). It is not illogical to think, then, that *FOX* genes, or some specific *FOX* genes anyway, may be responsible for development of neural structures.

FOX proteins are part of a much larger group of proteins called transcription factors. These control the genetic programs of cells by interaction with the regulatory regions of genes, which control how many copies of the gene's mRNA are made. The more copies, the more abundant the resulting protein will be in the cell (Marcus & Fisher 2003). The identifying feature of a *FOX* protein is the forkhead domain, which is the part of the protein that actually contacts the target region of another gene (Pinker 2001). As a transcription factor, *FOXP2* interacts with another gene to either activate or repress the transcription of the protein it makes. The exact genes with which *FOXP2* interacts and the resulting proteins of these interactions are still unknown at the current time, however one study did find that these interactions are able to repress certain lung-specific genes (Marcus & Fisher 2003). In any case, if *FOXP2* is mutated or is not in its correct form

for any reason, it will cause abnormal production of proteins. This could affect not only the genes that directly interact with *FOXP2* and their resulting proteins, but could also affect the genes with which the resulting proteins interact and this pattern can continue to affect numerous genes and proteins. These indirectly affected genes are called "cascades" (Marcus & Fisher 2003).

Normally, humans have two exact copies of each gene. It is likely that the difficulties that the affected members of the KE family experience are due to a mutation in just one of their copies. Evidence for this lies in the fact that *FOXP2* is found in other areas of the body: *FOXP2* plays a role in the development of the lungs, heart and gut during embryonic development. If both copies of the *FOXP2* gene were affected by the mutation in the KE family, it would have been less likely for certain organs to develop normally, which they did (Marcus & Fisher 2003). Tests confirmed that both the KE family as well as C.S., the unrelated individual with the break in chromosome 7, only had one disrupted copy of *FOXP2* (Pinker 2001).The idea that only one copy of *FOXP2* is damaged suggests that it is not the disruption of the gene that causes the language disorder, but the lack of two normal *FOXP2* genes that disallows normal development (Marcus & Fisher 2003). Thus, at the point in fetal development critical to the development of linguistic systems, only half of the transcription factor is present that is necessary for normal brain development (Pinker 2001). Another possibility is that there are slight differences in the lungs, heart and gut of the KE members due to the mutation in *FOXP2* (Marcus & Fisher 2003), however, it is difficult to study the subtleties of an individual's organs in a manner that allow these differences to be found.

Clearly, something went wrong in the development of some neural structures required for language in the brains of the affected members of the KE family. One place where this could be is in the cerebral cortex. This cortex is made up of about 6 layers, although there are not actually any boundaries between the layers. The inner layers develop first. In the inner three layers of the cortex, there are pyramidal cells, which are responsible for communicating with the other areas of the brain. Broca's area, which is an area of the cortex that is strongly correlated with language (and is discussed in more detail later in this paper), contains noticeably large pyramidal cells in its inner layers suggesting that the area communicates with other areas in a unique way. Furthermore, *FOXP2* is found only in the inner three layers, suggesting that the gene's important period in the developmental process is earlier in the development of the cortex rather than later and thus, in the development of Broca's area as well. Should *FOXP2* fail to trigger the proper conditions, as might be the case with the mutant *FOXP2* that is found in the affected KE family members, there would be implications for the connections between the cortex and other brain areas as well as the composition of the cortex itself. This hypothesis would also explain why there are lower levels of gray matter, which is brain matter responsible for stimulus-response activity, in the limbic system of the affected KE family members (Cooper 2006).

While a disturbance in *FOXP2* may necessarily lead to a language disorder or other developmental difficulties, the diagnosis of Specific Language Impairment is not sufficient to conclude that the individual has a genetic mutation. Most forms of SLI are not as severe as that of the KE family and it is not uncommon for children to have a less severe form of SLI or other general language impairment. Most of these disorders are

likely to be caused by a number of genetic and environmental factors combined (Maeburn et al. 2002). Maeburn and colleagues genotyped 270 language-impaired children (whom they had tested on a battery of linguistic tasks), thus, 540 alleles, and did not find even one guanine to adenine nucleotide mutation (2002). The researchers emphasize heavily that single-gene disorders are rare and severe, but more common disorders are generally caused by multiple genetic and environmental factors. Therefore, the KE family and C.S. are an exception to the norm and researchers should not expect to be able to use *FOXP2* as a means for examining general language disorders.

In sum, the language impairments of the affected members of the KE family in addition to the physiological information from C.S. allowed the family's inherited disorder to be traced to the *FOXP2* gene. Once the gene had been found, researchers could start searching for the genetic basis of the disorder. Based on knowledge of the functions of other *FOX* genes, it is likely that *FOXP2* plays a role in the development of neural structures required for language. It should also be noted that most people with language disorders have a normal set of *FOXP2* genes and therefore, *FOXP2* should not be held responsible for language disorders generally, however, it is necessary for a person to have two normal *FOXP2* genes in order for the neural structures required for language to develop normally.

FoxP2 AND ITS ROLE IN VOCAL BEHAVIOR

Much of the support for the evolutionary importance of *FOXP2* in distinguishing humans from other species comes from research involving animals. First, there is evidence from research done on birds, our closest vocal-learning relatives (Haesler et al. 2004), showing that *FoxP2* plays a significant role in their vocal-learning behavior as

well. This provides support for *FoxP2* generally being linked to vocal behavior in one way or another. Also, the patterns of changes that have occurred in the structure of *FoxP2* in other species, specifically non-human primates, taken with the changes in humans suggest that the gene was positively selected in human evolution. This indicates that over time, there was evolutionary pressure for the gene to mutate in the way that it did. The findings of these areas of research demonstrate support for the evolutionary importance of *FoxP2*.

***FoxP2* in birds**

In order to show that studying *FoxP2* in birds is useful to making conclusions about *FOXP2* in humans, it is necessary to first establish the similarities between avian communication and human communication. Both birds and humans communicate using combinations of phonemes put together in ways that can be either syntactically correct or incorrect. Both birds and humans have the ability to learn a second song dialect or language, respectively (Neapolitan, Pepperberg, & Schinke-Llano 1988). Furthermore, there are striking similarities in the acquisition process for both first and second languages/song dialects, as the case may be. Pre-linguistic humans and birds both participate in babbling behavior before they produce the words or song pieces of their native language or song repertoire (Snowden, Elowson & Roush 1997). Both humans and birds, specifically the white-crowned sparrow in a number of studies, have a sensitive phase, if not a critical period, for acquiring "nativelike phonology." This means that it would be significantly harder to learn the phonological rules if they were not learned during this period. Also, neither humans nor birds can learn a second language or song dialect without input that has a clearly demonstrated function and relevance and that is

comprehensible (Neapolitan, Pepperberg, & Schinke-Llano 1988). Thus, it would not be sufficient for either a human or a bird to learn from an audiotape because the input would not be functional or relevant to the learner without a context for the vocalizations. So simply being exposed to a new language or song dialect does not cause the listener to learn it.

Three independent groups on the avian lineage have developed vocal learning behavior at some point in their evolution—songbirds, parrots and hummingbirds. All of the birds in the three groups must learn at least one aspect of their songs by imitation of more experienced singers of their own species (Scarff & White 2004). The fact that vocal learning developed independently on three occasions in the avian lineage implies that there is some underlying factor common to birds that causes some birds to develop this behavior. Note that not all birds that vocalize are vocal learners; some birds have innate noises or songs that they will make regardless of whether they heard the sounds from other birds of their own species or that can be learned through simple imitation of other birds (Scarff & White 2004). Thus, there is no phonology to be learned or productive creativity that can be used. *FoxP2* is shown to be linked to vocal learning behavior in songbirds. Many of the studies regarding avian vocal learning have been done with zebra finches because male zebra finches exhibit vocal learning behavior by acquiring a courtship song while females do not. Thus, the brain regions related to singing in zebra finches is present in males, but smaller or even completely lacking in females (Teramitsu et al., 2004). This makes zebra finches a good species to study because there is a song-learning and a non-song-learning population within the same species, facilitating accurate comparisons between the two groups. A number of studies

that have been done with zebra finches show that the expression *FoxP2* in these birds is associated with singing behavior.

Researchers have found that *FoxP2* expression is found even in the embryonic zebra finch brain, however this expression is found in non-vocal-learners as well as vocal-learners (Haesler et al., 2004). Thus, it is not the presence of the protein itself responsible for the singing behaviors. In fact, there was expression of the gene found in the striatum and the dorsal thalamus, areas of the brain, in all of the 11 different species of birds studied and even in the crocodile that was studied as the closest non-avian existing relative of birds. Within the dorsal striatum, birds have a nucleus that is part of the song system, known as *Area X*. Area X in songbirds is part of a forebrain pathway that is mandatory for vocal learning. It has been found that lesions in this area during vocal learning lead to more plasticity in songs, thus it is thought that Area X helps to maintain song stability.

In male zebra finches, Area X shows higher levels of *FoxP2*'s protein than the surrounding areas in the striatum 35 to 50 days post-hatch, an age at which the birds are learning to produce song. There was no difference in the protein levels before or after this period (Haesler et al., 2004). This provides further evidence for a critical period between hatching and adulthood for the acquisition of singing behavior. For white-crowned sparrows, this period is 10 to 50 days post-hatch (Neapolitan, Pepperberg, & Schinke-Llano 1988). This hypothesis is supported by research on song acquisition in white-crowned sparrows. The birds tutored via audiotape in their natural song dialect during the sensitive period produced the song, but when the tutoring occurred after the sensitive phase, the vocalizations of the birds were similar to those of birds raised in

acoustic isolation. Furthermore, when it comes to second song dialect acquisition, sparrows that are tutored during their sensitive period create more exact copies of the song than sparrows that are tutored later or after this sensitive period (Neapolitan, Pepperberg, & Schinke-Llano 1988). As a result of these findings, we have reason to believe that a sensitive or critical period does exist in avian vocal-learners.

Not all birds demonstrate the same period for higher *FoxP2* expression or for song learning. In canaries, it was found that levels of *FoxP2* in Area X varied not by a critical period depending on development but by season of the year. During the season when the birds add new syllables to their singing routines and when the songs are more likely to be inconsistent, higher levels of *FoxP2* were observed in Area X in comparison to the surrounding areas. During the seasons where the song is static, particularly during breeding season, there was no significant difference found between the levels of the *FoxP2* protein in Area X than in the surrounding areas (Haesler et al. 2004). Across species, these differences in *FoxP2* expression correspond to the state of song plasticity (Scarff & White 2004). This may still provide further evidence for a critical period for song acquisition in birds, however, points out that the concept of "critical period" could differ from species to species. For example, the vocal learning period in humans and zebra finches seems to be a developmental phase not long after birth, but for canaries the vocal learning period cycles with the seasons (Haesler et al. 2004). It could be purported, then, that there is some mechanism which triggers the *FoxP2* protein levels to increase and this in some way influences the appropriate areas of the brain to be receptive to vocal communicative behavior.

Assuming that some type of critical period does indeed exist in song-learning birds, it is possible that the elevated levels of the *FoxP2* protein in zebra finches could be responsible for this sensitive period in which the bird learns its songs. It should be explored to determine whether or not *FOXP2* in humans could be connected with the critical period for language acquisition as well. As will be discussed in greater detail below, there is evidence that *FOXP2* had evolutionary effects on language. Therefore, it is plausible that there could be some sort of influence of *FOXP2* in humans on the biological structures and mechanisms underlying this period, just as *FoxP2* could potentially be underlying similar structures in songbirds. Another interpretation of this connection between *FoxP2* and a sensitive period in birds could be that a third variable is causing both the elevated levels of the *FoxP2* protein and also the sensitive period. If so, this finding could also be applied to humans and be useful in understanding the process of human language acquisition.

While it is probable that *FoxP2* has some specific relationship with song learning, claiming that elevated levels of *FoxP2* are responsible for song learning should be done with caution. Although other species of songbirds, such as chickadees and strawberry finches, do indeed exhibit the same correspondence between Area X and *FoxP2* that was mentioned above: levels of the protein are higher in Area X than in the surrounding areas. However, in song sparrows and Bengalese finches, levels of the *FoxP2* protein were lower in Area X than in the surrounding regions (Haesler et al. 2004). One might argue that this is the case due to varying levels of vocal syntax complexity, as Haesler and his fellow researchers originally did. They explored this possibility and found that the pattern of complexity across the species varied in a way that did not correspond to the

relative levels of *FoxP2* in Area X, and thus, vocal syntax complexity cannot account for these differences (Haesler et al. 2004). There could be some influence of the breeding season. It was found in some of the birds that *FoxP2* is not increased during breeding season in Area X, while expression of *FoxP2* during times not within the breeding season is higher (Haesler et al. 2004). Possibly, higher levels of the protein are for some reason inhibited during the breeding season.

Another possibility that was not mentioned by Haesler et al. regarding the reason that some species do not have elevated levels of *FoxP2* in Area X is the potential differences in the innate components of birdsong. In some species, the birds tend to sing the same song or same few songs across the entire species and will learn to sing that song with little variation even if raised without the presence of other birds of the same species, because they have innate repertoires of songs (Neapolitan, Pepperberg, & Schinke-Llano 1988). Other species of birds learn new songs regularly (Haesler et al. 2004). Potentially, the birds who do not display these elevated levels of *FoxP2* in Area X relative to the surrounding areas may have songs that are more "hard-wired." Therefore, the songs and calls that they sing are mostly biologically-based and there is very little in the way of learning. The extent of such learning could be simply imitating the songs of others and not learning all of the different components of the songs and ways that these components can be strung together properly for that species of bird. If this is true, then there is even further evidence for the importance of *FoxP2*'s relationship to vocal learning and possibly a critical period.

Furthermore, since it is thought that Area X helps to maintain song stability in birds and there are higher levels of *FoxP2* in Area X when it is most plastic for song

learning, then *FoxP2* could potentially be repressing proteins that cause the stability (Haesler et al. 2004). If this is the case, perhaps the gene works in the same way in the human brain. Maybe there are higher levels of *FOXP2* in areas related to language during the critical period for language acquisition. Indeed, *FOXP2* expression was found to be higher both in intensity and extent within a developing fetal human brain (Lai et al., 2003), but in order to study the levels of children, it would be required to have actual brains or parts of brains of children, which may prove difficult to obtain[**]. However, if there are high levels during the children's critical period for language acquisition, then *FOXP2* may be causing repression of the genes that keep the human brain stable, specifically the areas related to language. Therefore, *FOXP2* would be causing the critical period to occur.

***FoxP1* expression in birds and humans**

FoxP1 is the most closely related gene to *FoxP2*, and therefore, it is possible that their functions and expression are similar. *FoxP1* was expressed in all of the songbirds studied, but in zebra finches, *FoxP1* was found to demonstrate a sexually dimorphic split that corresponded to the dimorphism of the song circuit (Scharff & White 2004), which is that males are vocal-learners while females are not. In human embryos, *FOXP1* and *FOXP2* were found to be expressed in patterns highly similar to one another. The two genes also exhibited overlap in the thalamus, which has strong connections to motor and premotor cortex. Both genes express a pattern consistent with the idea that they are involved in sensorimotor integration, which is important to both vocalization and complex learned motor movements (Scharff & White 2004). Teramitsu et al. (2004)

[**] Actual brains are needed for this type of research as opposed to research that simply looks at the *FoxP2* gene because this type of research actually measures the levels of the FoxP2 protein in the brain.

suggest that *FoxP1* and *FoxP2* could act as coregulators in the brain. Although there is not sufficient data regarding *FoxP1*'s possible relationship with language, there is some evidence that it is likely to play a role in the formation and function of articulatory circuits in both humans and birds (Teramitsu et al. 2004). As described above, *FOX* genes tend to play a role in developmental complexity, so given their overlap in location, it is entirely plausible that both *FoxP1* and *FoxP2* are significant factors in the development of areas required for vocal communication.

Thus, studying *FoxP2* in avian vocal learners and non-learners can give us further insight into how *FoxP2* relates to vocal language in humans. Because levels of *FoxP2* are elevated in areas of the brain associated with singing behavior in vocal-learning birds, we can conclude that there is a clear connection between the levels of *FoxP2* and vocal-learning behavior. Also, this finding allows for a hypothesis that *FoxP2* is related to the critical period for language acquisition at least in birds, but the relationship may be present in humans too. Also, through studying birds, *FoxP1* has been indicated as another possible gene to study regarding vocal behavior. It has been found that the two genes tend to overlap in location as well as function in the brain. Because there are so many similarities in the vocal behavior of birds and that of humans, it is relevant to question whether *FoxP* genes have very similar functions in both birds and humans.

FoxP2 AND THE EVOLUTION OF LANGUAGE

FOXP2 is one of the most highly conserved genes—or least changed genes—when the genes of humans and rodents are compared: it is among the top five percent of highly conserved genes. The protein is 98% identical in the zebra finch (Cooper 2006), the bird discussed in the studies above. In the chimpanzee, gorilla and rhesus macaque,

the *FoxP2* proteins are all identical to one another, but contain two differences from the human protein and one from the mouse (Enard et al. 2002). This provides evidence for the claim that there was a high mutation rate of the *FOXP2* gene in humans since two of the three differences in amino acids from the mouse have occurred since the human and chimpanzee lineages diverged approximately 70 million years ago. The mutation rate is considered to be high because two changes must have occurred in the time period during which only one change would have been expected to occur. One of these changes may have had extreme functional consequences because it created a possible target site for phosphorylation by protein kinase C, a process which when occurring to forkhead transcription factors is crucial to mediating transcriptional regulation (Enard et al. 2002), or the amount of protein produced by genes. Enard and his colleages found that the pattern of variation of the amino acid amongst humans demonstrated a selective sweep (2002), meaning that the mutation increased the "fitness" of its carrier and increased the carrier's chance of survival and procreation. The reasons for the fitness of those with language will be discussed below, after it is established that the mutation in *FOXP2* was indeed crucial to the development of language. First, it is important to discuss the primary centers for language in primates and how they may have evolved into the human forms, which are capable of the complex linguistic systems known to modern humans.

Language areas in primates

Broca's area is usually regarded as one of the critical language centers in the brain. People who experience damage to Broca's area tend to speak telegraphically and laboriously, using mostly content words. They also have trouble comprehending phrases with complex word orders and repeating long words accurately (Cooper 2006). The

mutation in the KE family produces symptoms similar to Broca's aphasia, but Broca's area is not exclusively affected by the mutation (Vargha-Khardem et al. 2005). Because humans are the only primates that use vocal communication extensively, Broca's area must have developed into a speech center at some point during human evolution.

Broca's area in humans is homologous with similar regions in other primates (Cooper 2006). The homologue for Broca's area in great apes displays left-over-right asymmetry, similar to that of humans (i.e. most humans and great apes tend to use the right hand and foot more because of left-hemispheric dominance). This left-hemispheric dominance in Broca's homologue is also seen in other primates as well as early humans (Cooper 2006), implying that even if early humans did not have a Broca's area dedicated to language, the homologue was physically present. From this, we know that homologues of Broca's area in other primates are used for functions other than language, and thus, the development of hemispheric asymmetry was not the causative event in the emergence of language. While it might be tempting to try to make broader assertions about the role of *FOXP2* in the lateralization of the brain, this should be done with caution. Scarff and White found that *FOXP2* expression in human embryos of 19 to 22 weeks of gestation was not lateralized (2004), implying that either the expression was measured prior to lateralization or mechanisms downstream of *FOXP2* induce lateralization. This does not completely rule out the idea that *FOXP2* could have some responsibility for lateralization of areas in the brain, since the embryos studied were not very old. Furthermore, *FOXP2* could interact with other features later in development to play a role in lateralization, however, it should be noted that there is no concrete evidence for how or if this actually occurs.

In the monkey, Area F5 is thought to be the primate homologue of Broca's area (Corballis 2004) and is also an area of the monkey's brain where mirror neurons have been identified. Mirror neurons are a special type of neuron found in primates, including humans (according to Cooper 2006), that fire the same groups of neurons when the animal performs an action in addition to when they observe the same action being performed by others (Corballis 2004). Actions and gestures are important for social animals (Cooper 2006). Humans also have a motor system with mirror properties (Rizzolatti & Craighero 2004). By recording motor-evoked potentials (MEPs) it has been shown that the right hand and arm experience stimulation while participants observed grasping gestures and meaningless gestures (Rizzolatti and Craighero 2004). Also, when observing an action, there is an inhibitory mechanism that prevents the person from actually performing the action, even though there is activation of the same neurons that would cause performance of the action. This inhibition allows the cortical motor system to reaction to the observed action without necessarily performing any overt movement (Rizzolatti & Craighero 2004). Researchers believe that mirror neurons provided a biological foundation upon which language evolved and that mirror neurons also support the hypothesis that language evolved from manual gestures instead of the vocalizations of our primate ancestors (Corballis 2004).

The discussion of gestures inevitably brings up the question of how sign languages fit into the evolution of language. Humans can learn language from any sensory modality where there is appropriate linguistic flux, but language does not depend on any particular mode of sensory input (Ruben 2005). Ruben claims that language is an intrinsic property of the central nervous system (2005), so sign language and spoken

language are equally easy to learn, so long as there is meaningful sensory input. Furthermore, sign languages are no new invention; Socrates references the use of signs by those who are deaf in the 4th century BC and a Greek vase from the 5th century BC depicts a woman whose tongue was cut out using signs (Ruben 2005). Therefore, sign language can be thought of as being the same as spoken language in terms of complexity and acquisition process, generally, except that different physiological structures are required for each language.

One line of evidence for the theory of linguistic evolution that claims that spoken language evolved from gestural language is that infants use manual gestures before using vocalizations (Stokoe 1978). Infants use pointing and reaching first before even accompanying them with vocalizations, and after they do start to vocalize, they vocalize simultaneously with the gestures (Stokoe 1978). Even adults use gestures to reinforce their own verbal communication, so manual gestures clearly play a necessary role even in spoken language. Futhermore, Stokoe claims that there is evidence that gestural signs are easier for children to learn, particularly when the signs are iconic (1978), meaning that they are not arbitrary. This is supported by the fact that autistic children find referential signs and signs about emotions easier to understand and can these signs can be understood earlier than speech. Another area that supports the idea that gesture is easier for children is that deaf children of hearing, non-signing parents develop a syntactic patterns in their gestures, even though the gestures made by their parents in an effort to communicate with them do not have these patterns. This provides evidence that humans had the drive to label objects and events with gestural signs and then later developed a syntax for constructing meaningful phrases with the signs. Also, that gestural signs occur

before vocalizations get coupled with them rules out the possibility that there was a separate parallel development evolutionarily. From the fact that the grammars of sign languages cannot have derived from corresponding parts of spoken language grammars, it can be determined the notion that sign language developed from spoken language is incorrect (Stokoe 1978). Evidence from the KE family suggests that *FOXP2* may be associated with sign languages as well. The deficit in the perception and production of rhythm and timing that the affected members experience applies to both vocal and manual movements (Lai et al. 2003). Given the evolutionary implications of *FoxP2*'s association with sign languages, it is surprising that this is an area that has not been specifically researched.

To return to the subject of mirror neurons, the idea that spoken language developed from manual gestures is further supported by the pathway through which mirror neurons allow for this transition from one form of communication to another to occur. Mirror neurons create a direct connection between the sender and the receiver of a message because the same neurons are firing (Rizzolatti & Craighero 2004). In other words, the neurons that are activated when the sender is performing the action are the same neurons being activated by the receiver who is observing the action. This provides a direct connection between the meaning of the message and the neurons that are being activated, creating a "nonabitrary, semantic link" between the two communicating individuals (Rizzolatti & Craighero 2004). Because mirror neurons in humans respond to pantomimes and meaningless actions, while mirror-neuron systems in monkeys do not, there is a transition already occurring just in the mirror-neuron system from a closed object-reference system to an open system allowing for communication about actions and

objects without direct reference to them (Rizzolatti & Craighero 2004). So, just by studying the mirror-neuron system, it is clear that the gestural linguistic system is more advanced in humans than it is in other primates.

Greater complexity in the gestural language systems of humans provides a base from which spoken language could have evolved. What is still somewhat unclear is specifically how hand and arm gestures transferred to spoken language with meaning. Rizzolatti and Craighero propose that manual hand and arm gestures and speech gestures share a common neural substrate (2004). These researchers hypothesize this based on data that people move their mouths more and speak at a louder volume when grasping a larger object as opposed to a smaller object. Furthermore, grasping also affects the volume and movements of the mouth when a person is observing another person moving a larger object. These findings demonstrate that hand and mouth gestures are linked in humans (Rizzolatti & Craighero 2004). This connection lays a foundation for spoken language: a sound may come from the way an individual moves his mouth based on the object to which they are referring or the action that they are performing and eventually, the action or the reference may no longer be needed by the conversational partners to understand the meaning of the sound (Rizzolatti & Craighero 2004). Further evidence for this process stems from the fact that the mirror neurons appear to activate for goal-oriented movement of the hand and mouth in other primates as well as in humans (Rizzolatti & Craighero 2004). Thus, although our nonhuman predecessors may have been in some way capable of understanding the communicative importance of goal-oriented hand and mouth gestures, they were not capable of developing this system into spoken language. This was due to a lack of the correct anatomical structures and also of

the correct neural structures, which could have been changed in humans due to changes in *FOXP2*, that were necessary in order to have vocal language. Furthermore, Broca's area itself in humans has been associated with coding for "meaningful" rather than "meaningless" gestures (Cooper 2006), although, as previously stated, neurons in the motor system do respond to meaningless gestures (Rizzolatti & Craighero 2004). Therefore, it is likely that human language evolved from gestures and that mirror neurons played a large part in this development.

If, as demonstrated by Enard et al. (2002), *FOXP2* was positively selected in the past 200,000 years and was selected specifically for the development of language, as hypothesized in the present paper, there is a high probability that *FOXP2* influenced the development of the speech and language centers in the brain. Corballis (2004) suggests that *FOXP2* was responsible for the recruitment of Broca's area for language, as it was necessary for this area to develop in order to deal with the complex functions of syntax. Corballis goes on further to imply that *FOXP2* was responsible not only for recruiting Broca's area for language, but for recruiting it for speech (2004). It is possible that the use of Broca's area for language is natural considering the evidence from the mirror neuron system of other primates. However, these primates lack the capabilities of speech for a variety of reasons (discussed below). *FOXP2*, thus, may have been a factor leading to humans having the brain capacity for vocal communication. More support stems from the ability of primates to be taught how to use syntax in a productive manner, in other words, to use their knowledge of syntax rules to produce statements with sign language, gestures or other symbols. These "languages" fall short of a fully syntactic language, but nonetheless, Corballis believes that their presence demonstrates that syntax may have

been present prior to vocal communication and concludes that *FOXP2* may have been responsible for "perfecting" speech as a medium of communication (2004).

Bosman et al. (2004) think that researchers should be cautious in making claims such as Corballis's. They argue that in the Broca's area homologue in monkeys, there are neurons similar to mirror neurons that respond to vocalization and other acoustic stimuli. The researchers hypothesize that the neurons are part of a vocal mirror system used for vocal imitative behavior (Bosman et al. 2004). However, they do not mention that these neurons actually fire when simply hearing a sound. Corballis (2004a) points out that if there are mirror neurons involved in vocal behavior, it is because the neurons are stimulated by the physical appearance of the primate creating the sounds. A mirror neuron system for vocalizations themselves has not been demonstrated by any study. The suggestions of Bosman et al. would have implications on the knowledge about *FOXP2* because they suggest that rather than impacting the mirror neuron system, it is the working-memory system that is affected by the gene (2004). They support this by citing the impairment in non-word repetition of the KE family, a condition that is normally considered to be a deficit in phonological storage in working-memory (Bosman et al. 2004). These two theories do not have to be mutually exclusive. As suggested by Bosman et al. (2004), there could be anatomical and functional overlap between the working-memory system and their proposed vocalization mirror system. Even if there is no vocalization mirror system and the mirror neurons are firing based solely on visual perception of gestures, as Corballis claims, it is still likely that there is overlap between the mirror neuron system and the working-memory system, as both seem to be imperative parts of human language acquisition mechanisms.

There is evidence that the *FOXP2* gene was positively selected in evolution around the time that human language developed: the two changes in the gene occurred rapidly at around twice the expected rate of mutation. This is indicated by the calculation of mutations occurring in a certain period of time which is done by studying the number of mutations in each species and calculating the time period in which they occur. On the human lineage, two mutations have occurred, when only one would have been expected based on the numbers of mutations in the gene in other species during similar amounts of time. This rapid change indicates that there was an intense evolutionary pressure at the time. These changes and their association with the positioning of the alleles on the seventh chromosome, there is evidence of an evolutionary "sweep" pointing to important changes related to Broca's area and language (Cooper 2006).

One might ask what type of evolutionary pressure would lead to the development of vocal communication. Corballis is one of many researchers who posit that language in the form of gestures was present before vocal language (2004b). Other primates, and likely ancestors of humans, lack the physical structures required for vocal communication, such as a bent vocal tract or specific proportions and fine motor control of the mouth and tongue. Evidence exists that the lowering of the larynx, which is also required for human speech, was not a complete adaptation in Neanderthals about 30,000 years ago. This incomplete adaptation that would have led to poor articulation would probably have been enough to put them at a lagging social position compared to *homo sapiens*, which may have led to their eventual distinction (Corballis 2004b).

Corballis believes that autonomous speech was carried by *homo sapiens* that emigrated from Africa about 50,000 years ago (2004b), however, he does not mention

what evolutionary pressure caused this. It could have been the emigration itself. The *homo sapiens* were traveling and were likely to be carrying a number of things with them. Thus, it would not have been possible to use their hands to gesture in the ways they had previously used to communicate. Corballis says that it could have been simply a cultural invention, "born of the discovery that language could be accommodated entirely through voiced articulatory gestures and decodable from the auditory signal alone" (2004b, pp. 547). This may be true, but it may be also that vocal communication was necessary and useful to the emigrants. Furthermore, there were even earlier exoduses from Africa that may have started the changing of communication styles from manual and gestural to vocal about 100,000 years ago (Corballis 2004b). Since it is expected that mutation in *FOXP2* occurred somewhere near this time period, there may have been environmental pressure (i.e. the need to travel) for the mutation to occur. The consequence of these changes allowed humans to further emigrate and spread, causing these linguistically better adapted creatures to dominate in a larger area.

One problem with proposing that a single genetic event caused language is that it is then necessary to conclude that all subsequent *Homo sapiens* from which modern humans have their origins can trace their ancestry back to one individual (Crow 1998). That is to say that the lineages of all *Homo sapiens*, other than the individual in whom the mutation occurred and those directly descended from that individual and carried the mutation, were too unfit to survive into modernity. This implies that there was something truly remarkable about the mutation in *FoxP2* and thus, as proposed here, spoken language that caused those who possessed this mutation to be more fit to survive. But why does language make humans more fit to survive? Pinker and Jackendoff argue that

without language, there are human concepts that are not learnable without language (2005). These include knowledge about periods of time, mental states of other people, social roles and various other concepts that are taken for granted in human life. The hypothesis here is that language reflects these uniquely human structures that are not necessarily language-based systems, but rely on linguistic expression (Pinker & Jackendoff 2005).

Language was developed because it became necessary for people to communicate thoughts and ideas with other people. As mentioned before, this may have been due to a migration, in which people would have needed to share ideas in order to efficiently make such a massive migration in a group. The alternative view is that language was designed specifically for thought, however, this is unlikely because there have been documented cases of language emerging spontaneously in groups of people without previous linguistic knowledge[††], but there are no documented cases where language was just developed for internal monologue or thought (Pinker & Jackendoff 2005). Futhermore, people who have not learned any language for one reason or another (in many cases, these are deaf people who were not discovered to be deaf until a few years after birth) still possess the ability to reason (Napoli 2003). Thus, having thought is not dependent on language, although the understanding of some complex concepts may be facilitated by language (Pinker & Jackendoff 2005). Since we know that language developed as an adaptation to communicate about knowledge and intentions, it is not difficult to reason that there was evolutionary pressure for language development, and thus mutations in *FOXP2* that facilitated language to survive. It is plausible that those with language were

[††] A community of deaf people in Nicaragua invented a sign language in order to communicate with one another (Pinker & Jackendoff 2005).

more likely to be able to adhere to the social requirements of the group and not be isolated or left behind during the migration, while those without language tended to be left behind or not warned effectively about certain dangers or perils throughout the migration. In this way, language was particularly adaptive.

In sum, the present analysis proposes that it was necessary to express knowledge and intentions to others during a migration of early *Homo sapiens*, meaning that language was necessary. A mutation that occurred in *FOXP2* allowed for the correct neural structures to develop, possibly recruiting Broca's area for linguistic usage. With these neural structures intact, language could begin to be built amongst those who had them. Eventually a social structure developed and those who participated in the society by using language were more likely to survive than those who did not have the linguistic ability to participate in it. Therefore, the mutation in *FOXP2* was an adaptive change for survival.

***FoxP2* and a critical period for language acquisition**

There is a possibility that *FOXP2* is connected both biologically and evolutionarily to the critical period of language acquisition[‡‡]. Scarff and White conclude that *FoxP2* may be an ancient transcription factor that creates and maintains sensorimotor circuit in such a way that when the conditions are right, a permissive environment for vocal learning is created (2004). This could explain while vocal learning is not present in all species that exhibit *FoxP2*: the conditions were not right either biologically or environmentally for a permissive vocal learning platform. This conclusion can also help to explain in what ways *FoxP2* may be related to a critical period for language

[‡‡] It should be noted that the critical period hypothesis (which suggests that neurological changes in the brain occur around the time of puberty which make learners of language less capable of acquiring a language) has not been demonstrated unequivocally to be correct (Carroll 2004). The author of the present paper bases arguments in this section on the assumption that it is indeed correct.

acquisition. If *FoxP2* is one of the factors necessary for a permissive vocal learning environment, the elevated levels that were found in birds may be indicative of some other regulatory factor interacting with the gene to create this period of plasticity for vocal learning. It is likely that there is a similar occurrence in humans, however, no research regarding *FOXP2* has been done on the brains of children during the critical language acquisition period. Thus, it is necessary to look beyond *FoxP2* and more broadly at the factors involved in the critical period for language acquisition to gain an understanding of how *FoxP2* may be involved.

To begin with, there is little evidence of a critical period in nonhuman primates, due to the fact that primates are not linguistic-"learners". Many of the communicative vocalizations and gestures seem to be genetically innate in nonhuman primates, although they do need to be perfected by experience (Seyfarth & Cheney 1997). One line of evidence for this is that cross-fostered monkeys, such as Japanese macaques fostered by rhesus macaques, produce the calls made by their own species' as opposed to those of their foster species. However, the amount of vocalizations made by the fostered monkeys was somewhere between the usage of their own species and their foster species (Seyfarth & Cheney 1997), implying that some experience is necessary to determine the appropriate usages for the calls. Furthermore, Seyfarth and Cheney state that these two particular types of monkeys have the same physiological structures, so the differences in vocalization are not due to physiological differences in the species (1997). Another line of evidence for the hypothesis that nonhuman primates have more innate aspects to their communication than humans is that most vocal calls are nearly fully-formed shortly after birth. There is little developmental progression in the vocalizations. An adult-like, but

somewhat imperfect, call may be used only days after birth, but will not change until a few months of age to a year (depending on the species of monkey), when the call suddenly is heard in its final, adult form (Seyfarth & Cheney 1997). Thus, because these nonhuman primates have innate sets of calls, there is no critical period necessary to learn vocalizations, however, the perfection of the calls and the usage seems to be set during the first year of life. Of course, this does not mean that the period during which calls are perfected is a "sensitive" or critical period; it is more likely that the perfection of calls occurs during the period after birth because the animals are exposed to the adult forms of the calls from birth, if not *in utero*. However, it would be an interesting study to raise some vocalizing monkeys in complete isolation for the first year of their lives and then expose them to the calls of adults of their own species. If the monkeys failed to perfect their calls or had a markedly more difficult time doing so, it would provide evidence for a critical period for the maturation of vocalizations.

Humans demonstrate a "storage" phase beginning in the prenatal period, where children become attuned to the vocalizations of their mother and lasts until children begin to speak words in their native language. In this period, they are storing the utterances they hear and beginning to make sense of them. By the age of ten months, normally developing human infants can respond appropriately to about 67 words (Locke & Snow 1997). Nonhuman primates do not demonstrate this period typically, as there is no need to store communicative vocalizations when the vocalizations are innate, however, Locke and Snow do note one case where storage of spoken words was stored by one bonobo named Kanzi. Kanzi was not specifically taught the meanings of the words, but acquired them from being part of a human-like social environment (Locke & Snow 1997). Even

though Kanzi participated in what seems like a period of storage at an appropriate age, since his case in an isolated occurrence, there is no way of knowing whether the storage period was due specifically to his age (and thus, a critical or sensitive period) or due to other factors, such as the unique environment.

To summarize the findings regarding primates and a critical period for language acquisition, there is no significant evidence for a critical period in nonhuman primates, although it has not been completely ruled out either. This is mainly because the majority of vocalizations in nonhuman primates seem to be genetically predetermined: infant primates can make the vocalizations of their species effectively, but not perfectly, very shortly after birth. Nevertheless, it is possible that there is a critical period, possibly *in uetero*, for the primates to learn the vocalizations or even just to perfect the vocalizations. Perhaps if the primates do not perfect their calls during a certain period when the communication centers in their brains are malleable, they will not be able to perfect them later in life. This is somewhat unlikely considering the fact that pygmy marmosets have been found to be capable of changing their call structure at any time in their development, suggesting a more open vocal development model as opposed to a closed critical period model (Snowden, Elowson & Roush 1997). These types of changes are made because of social influences and occur most often when the social environment is undergoing change. The researchers note that all of the calls themselves are present from a very young age and that no new calls are learned (Snowden, Elowson & Roush 1997).

If nonhuman primates do not undergo a critical period for language acquisition and *FoxP2* works in the way proposed in the present analysis, then one should expect to find no differences in *FoxP2* expression in the areas of the brain relevant to

communication relative to other areas of the brain during early childhood in primates. No study demonstrates this, but if this finding were true, there may be broader implications for *FoxP2*. If there is no critical period in nonhuman primates and also no increase in *FoxP2* in communication areas of the brain during early childhood, it would imply that the changes in levels of *FoxP2* found in birds are not just typical of development generally and that they are related to vocal learning. Furthermore, if the elevated levels of *FOXP2* were found in human children, it would indicate that the increase in levels is unique to vocal-learning creatures at the time when they are exhibiting vocal-learning. The above potential findings would therefore provide evidence that the critical period for vocal acquisition is indeed regulated by *FoxP2* levels in the brain.

Researchers in favor of the critical period hypothesis of language acquisition believe that there is a neurological change that leaves post-pubescent learners less able to learn language (Carroll 2004). This neurological change could potentially be a change in regulation of *FoxP2* in the primary language centers of the brain. If the *FoxP2* protein somehow regulates the plasticity of language areas like Broca's area in humans or Area X in songbirds, then it is possible that these elevated levels of the protein reduce during puberty. Perhaps *FoxP2* has some sort of timing mechanism that allows for its levels to be heightened for a period of time after birth and then slowly decrease throughout childhood. This would account for how the language centers of human children and songbirds that acquire songs within the post-hatch period remain plastic and receptive to language or song in the first part of life. However, it does not easily account for what happens in birds whose song acquisition abilities vary depending on the season or their

breeding cycle. Possibly, the timing mechanism is different or is in some way able to adapt to the particular needs of vocal learning of that species.

A relevant question to ask about all of this is why *FoxP2* would enable a critical period for vocal learning in humans and in songbirds, even though the two forms of the gene are different, but not enable a critical period in nonhuman primates, whose form of *FoxP2* is more closely related to that of humans? One possibility is that the mutations in the *FoxP2* gene of both birds and humans, even though completely unrelated, both resulted in elevated levels at specific times. Once the levels are elevated, there is no need to account for the differences between the human and the avian form of the gene: elevated levels of *FoxP2* could cause the language centers of the brain to be more receptive to learning. Another possibility is that it was simply adaptive at some point for both songbirds and humans to be linguistic/song-learners, and thus, the fittest to survive were individuals who had elevated levels of *FoxP2* and consequently, plasticity in the language centers of the brain. This would account for both nonhuman primates' inability to be linguistic learners and also for their lack of a critical period. A third possibility, of course, is that there just has not been enough research to show that nonhuman primates actually do have a critical period and are linguistic learners. In that case, they would presumably have elevated levels of *FoxP2* during their critical learning period.

Although this whole theory about the critical period seems incredibly speculative, I would like to point out the lack of research that has been done in this area. First of all, there is no research that has been done, except for on birds, investigating *FoxP2*'s affect on the critical period and the purpose of this research was not specifically concerning a critical period. Even in the studies demonstrating that levels of *FoxP2* in birds generally

correspond to periods of song learning, no researcher has previously suggested that the levels of *FoxP2* may have a causal relationship to the period or tried to account for ways in which they may be related. Also, there is not even any mention in the literature about *FoxP2*'s possible role in the human critical period for language acquisition or even how levels of *FoxP2* may vary in the life cycles of human and nonhuman primates. Thus, while the above may seem presumptuous, it is because there is a limited amount of research that has been done in the area of causation of the critical period for language acquisition, which itself is a controversial area. Nonetheless, given the evidence of *FoxP2* in avian vocal-learners it is hard to imagine that *FoxP2* is not related to the critical period, at least in birds, in a causal way. Therefore, the above discussion points out the necessity for research in the area of *FoxP2*'s relationship to the critical period in humans and also the need for further research in this area in nonhuman primates. Only then can we understand what may turn out to be truly the most fascinating consequence of the *FoxP2* gene.

There is, as demonstrated above, a likely connection between *FoxP2* and the critical period for language acquisition. This connection would tie the origins of both language generally, and the critical period for language acquisition together in evolutionary terms. Somehow, not only were those people who carried the mutated form of *FoxP2* more fit for survival because of their language abilities, but were also more fit because of the critical period for learning language when the levels of *FoxP2* were increased in linguistic areas. This hypothesis fits in with the idea that the increased levels cause the brain area to be more malleable and plastic, thus promoting learning.

Is *FOXP2* related to schizophrenia?

One of the major characteristics of schizophrenia is disordered language. Although it may seem like a stretch to say that *FoxP2* may be a cause of some symptoms of schizophrenia, researchers have suggested to the possibility in a few different contexts and thus, it merits a brief discussion here. Crow brings up schizophrenia in the context of language evolution in that it is a disadvantageous variation in cerebral asymmetry (1998). He claims that schizophrenia is a reflection of selective factors operating in the evolution of language. Therefore, suggests Crow, schizophrenia is "the price [humans] pay for language" (Crow 1998). Crow goes on to suggest that language and psychosis, such as schizophrenia, originate from a common source, which could in theory be related to *FOXP2*, and that the delusions and auditory hallucinations reflect a disorganization of language in the brain, namely, the establishment of cerebral dominance (1998). If this is true, based on the information regarding the human brain discussed above, it is possible that *FOXP2* could be in some way responsible for this disorganization in the brain, given its developmental role.

Another mention of *FOXP2* in schizophrenia research comes from research by Sanjuan et al. (2006). These researchers found through analysis of the DNA of psychotic patients that *FOXP2* might be specifically related to both the language disorder involved in schizophrenia as well as the disorder itself (2006). There was a significant relationship between certain haplotypes, or sets of gene sequences, on *FOXP2* and schizophrenia with auditory hallucinations. The researchers found that there was a relationship between the haplotypes and the frequency and duration of the auditory hallucinations during incoherent speech. Sanjuan et al. conclude that more research needs to be done in this area, but believe that there is indeed a relationship between *FOXP2* and the language

disorder as well as possibly the auditory hallucinations experienced by schizophrenia patients. Moreover, they conclude that *FOXP2* could be responsible for vulnerability to mental disorders with language impairments, such as schizophrenia and autism (2006).

One potential problem in the implications of these hypotheses about *FOXP2* and schizophrenia is that *FOXP2* seems to have a role in the development of neural structures, but the onset of schizophrenia typically does not occur until early adulthood. If something went wrong in the development of the neural structures causing language to become "disorganized" in the brain, as Crow puts it, then why would these psychotic symptoms not show up until later in life? However, the conclusion that *FOXP2* only influences the vulnerability to schizophrenia may be worthy of further study, given the fact that many times, onset of schizophrenia requires some type of environmental factor to cause the psychosis. Therefore, it could be that the organizations of haplotypes, as discussed by Sanjuan, produces a vulnerability but another environmental factor is needed for schizophrenia to occur.

FUTURE DIRECTIONS OF *FoxP2* RESEARCH

Although, as I have suggested above, a significant amount of research needed remains to be done to determine how *FoxP2* may affect the critical period for language acquisition, this type of research are far from simple. The difficulty is that it is necessary to examine actual pieces of the brain. While some *FOXP2* research enthusiasts may believe that the study of the gene is worth the sacrifice of numerous animals, I do not believe that this is ethical because there is little medical benefit to such studies. Remember that the mutation in the KE family is rare and severe and there are extremely few people in the world with this mutation. Thus, the study of *FOXP2* will never 'cure'

any language disorder and so the sacrifice of animals in *FoxP2* research cannot be justified by the benefit of curing a condition. Another problem is that human brains would be necessary to investigate the levels of *FOXP2* throughout the lifespan. Research using fetuses has been done regarding *FOXP2* in the past but only the early prenatal period can be studied through this method. Another possibility would be to use cadavers to do research on *FOXP2* levels, but in order to study the critical period, it would be necessary to obtain cadavers of children. Again, we must ask if there is enough medical benefit to studying *FoxP2* to warrant use of children's cadavers for it. That is not to say that there is nothing to be gained from studying *FoxP2*. There is benefit from it in that the research would add to the knowledge base about genetics and how the brain works. An excellent research development would be to find a way to study the levels of *FOXP2* in the brains of living organisms in a rather non-invasive procedure. This would make possible the study of the critical period hypothesis discussed above. Research on language evolution may be able to further explain exactly what types of genetic changes must have occurred in humans in order to have spoken language.

CONCLUSIONS

To conclude, a mutation in *FOXP2* could have been a crucial factor in the development of human vocal communication. There is strong evidence for this from the study of *FoxP2* expression in other animals and the proposed functions of it in the human brain. Not only has *FoxP2* been correlated with linguistic areas in the brain of birds, different levels of the protein produced by *FoxP2* are found in the areas based on whether the birds are learning songs. Also, the mutation chronologically fits into the time period where there was hypothesized to be evolutionary pressure for the mutation to occur and it

has been demonstrated that *FOXP2* was indeed positively selected during human evolution. Thus, the mutation in *FOXP2* led to the ability for language and language made its possessors more "fit" to survive, therefore, passing on the mutated *FOXP2* gene to others who were in turn, more "fit" to survive.

The study of songbirds has demonstrated that *FoxP2* is not only connected to vocal learning in general, but that it may be related, possibly even in a causal way, to the critical period in language acquisition experienced by vocal-learning animals. This hypothesis is more speculative than the others presented in this paper however, there is no reason to think that it is not a likely possibility considering the correlation between *FoxP2* expression in linguistic areas of the brain during song-learning periods in their lifespans. More research needs to be done in this field to determine whether *FOXP2* could be related to the critical period in humans.

Generally, there is much more research that needs to be done on *FOXP2* in order to fully understand its relationship with language in both a neurological and evolutionary sense. Further research however, presents an ethical dilemma because this type of research requires sacrifice of animals and use of human fetuses and cadavers, which are hard to come by. Because information about *FOXP2* will probably not ever lead to cures for any disorders or other medical benefits, the research would be simply for the sake of research on the gene. While this type of research is important and fascinating, researchers must decide whether using the required materials to do research not producing medical benefit is worth it, since the materials may be in high demand for various other types of research. Thus, while I hope research continues on *FoxP2*, the

evolution of language, and the critical period for acquisition, I hope that researchers use good judgment in their methods of research.

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